EXHIBIT 606.A

KEITH PATRICK GIBSON, PHARM.D., J.D.
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May 16, 2011

Don A. Ernst
Ernst Law Group
Attorneys at Law
1020 Palm Street
San Luis Obispo CA 93401

Re: McCornack vs. Actavis Totowa, LLC

Dear Mr. Ernst:

At your request I am providing my opinion on the post-mortem drug levels found in Mr. McCornack's blood, the distribution of digoxin and other drugs post-mortem, the effect of differing formulations of digoxin on bioavailability and the resulting clinical picture that is the likely cause of Mr. McCornack's death.

Daniel E. McCornack was a 45 year old Caucasian male who died on March 23, 2008 at approximately 0030. At the time of his death, he was 70 inches tall, approximately 220 pounds.[1] The Sheriff-Coroner's report from Santa Cruz County listed cardiac arrest as the cause of death.[2]

The records indicate that the deceased was taking the following medications:[3]

Drug	*Sig*	*Directions*
Diltiazem CD 360mg:	1 po qAM	One tablet every morning
Diltiazem CD 180mg:	1 po qPM	One tablet every evening
Digitek 0.25mg:	1 po bid	One tablet two times a day
Allopurinol 100mg:	3 po qDAY	Three tablets every day
Aspirin 325mg:	1 po qDAY	One tablet every day
Prevacid 30mg:	1 po qDAY	One tablet every day
Indocin	Prn	As needed

[1] Report Report of Autopsy Examination by Richard T. Mason, M.D., Forensic Pathologist
[2] Sheriff-Coroner, Santa Cruz County, Death Investigation Report and Supplemental Report, Case Number 08-02797, by N. Silva
[3] Refill Tracking Form: attached to the Deposition of Richard T. Mason, M.D., dated 10-01-2009

Kathy McCornack reports that her husband took his medication after breakfast and immediately after dinner every day. On the day of his death, Mrs. McCormack did not see her husband take his daily meds, however, she did have a conversation with one or both of her sons about Mr. McCornack's drug usage. The sons reported that their father consumed his medications immediately after the evening meal.

In her deposition, Mrs. McCornack describes the evening meal occurring in the late afternoon/early evening before sundown, or that the preparation for the evening meal began at that time. It seems the evidence then is that the deceased consumed his medications around 6 to 8 p.m and suffered a cardiac arrest at 30 minutes past midnight that same evening.

The following are the last ante mortem lab results for Mr. McCornack:[4]

Date	*Time*	*Digoxin*	*S Creat*	*Bun*
05-15-2007	0808	1.6	1.2	8.0

Post-mortem drug levels were obtained for alcohol, diltiazem, digoxin, quinidine /quinine and atropine.[5] Those levels were obtained from a peripheral site and the results are summarized below:

Drug	*Post-mortem Level*
Ethyl Alcohol	48 mg/dL (BAC = 0.48 % w/v)
Diltiazem	630 ng/mL
Digoxin	3.6 ng/mL
Qunidine/Quinine	Trace
Atropine	Positive

Mr. McCornack was prescribed digoxin to treat his heart condition.[6] Digoxin is a drug used to treat congestive heart failure and certain irregular contractions of the heart call arrhythmias. Digoxin can increase the force of myocardial contractions (positive inotropic effect) or can be used to treat irregular contractions of the heart known as arrhythmias such as atrial fibrillation, atrial flutter or ventricular tachycardia.[7]

The mechanism of action of the drug involves the increase in the influx of calcium ions from the extracellular to the intracellular cytoplasm of the cell. It does this by inhibition of the transport of sodium and potassium ion movement across the myocardial membrane. This increase in calcium ions results in a potentiation of the contractile force of the heart muscle fibers (positive inotropic effect). The drug also may inhibit adenosine triphosphatase (ATPase) and decrease conduction through the S-A and AV nodes.[8]

[4] Central Coast Clinical Lab, Templeton CA dated 05-15-2007
[5] Supplemental Toxicology Report of: McCornack, Daniel E., NMS Labs, dated 06-24-2011
[6] Deposition of Gordon Lemm, M.D., 10-02-2009; Deposition of Lawrence Von Dollen, M.D. 10-05-2009.
[7] Leikin & Palouchek's, *Poisoning & Toxicology Handbook*, 3rd Edition (2002), Pg 484
[8] Leikin & Palouchek's, *Poisoning & Toxicology Handbook*, 3rd Edition (2002), Pg 484

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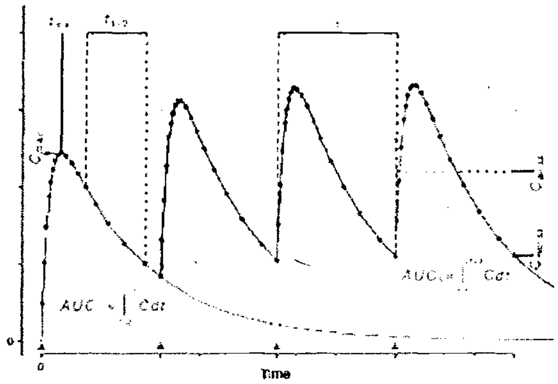
The distribution of the digoxin is minimal to body fat; high concentrations to myocardium, skeletal muscles and kidney; and it crosses the blood brain barrier. The volume of distribution in adults is 7 L/Kg (decreased with renal disease). The half-life in adults is approximately 39 ± 13 hours.[9]

Therapeutic digoxin levels are reported to be from 0.8 to 2 ng/mL.[10] Concentrations of digoxin above 0.8 ng/mL are associated with an inotropic effect.[11]

Toxicity is an important clinical problem. The therapeutic and toxic effects of digoxin are dose-related. The drug has a narrow therapeutic index. The therapeutic index is a comparison of the amount of a therapeutic agent that causes a therapeutic effect to the amount that causes toxicity or death.[12] Casually stated, therapeutic index is the difference between the therapeutic dose and the toxic dose.

Plotting the blood concentrations vs. time for a hypothetical drug is demonstrated by this graph. Where the blue line with the blue dots represents the blood level over multiple doses.:



Note the maximum level for the first dose is lower than the steady state peak level. The peaks and troughs for a drug will be consistent at steady state. Steady state is defined as that time in which the intake of drug equals the excretion of the drug. 90% of steady state will be achieved at approximately 4 times the half-life.[13]

When a new dose is introduced or the bioavailability is changed, the next peak after the change will be higher or lower depending on whether bioavailability increases or decreases. Thus a peak after a new larger dose will be higher than the proceeding peak and the peaks and troughs will be consistent once steady state is reached.

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[9] Gilman et al, *Goodman and Gilman's The Pharmacological Basis of Therapeutics*, 8th Edition, 1990; Pg 1675

[10] Ellenhorn M. and Barceloux D., *Medical Toxicology, Diagnosis and Treatment of Human Poisoning*, 1988, Elsevier; Pg 204

[11] Goodman & Gilman's The Pharmacological Basis of Therapeutics, 11th Edition , 2006

[12] *Katzung and Trevor's Pharmacology Examination & Board Review, 9th Edition*, McGraw Hill; Pg 15

[13] Evans W.E., Schentag J.J., Jusko W.J., *Applied Pharmacokinetics: Principles of Therapeutic Drug Monitoring* (1980); Pg 342

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Digoxin is available in two strengths as a tablet: 0.125mg and 0.25mg.[14] Bioavailability is the extent to which a drug becomes available to the target tissue after administration.[15] Gastrointestinal (GI) absorption of digoxin is normally approximately 60 to 85%.[16] [17] It can be as high as 100% with some formulations.

The onset of action from an oral dose is 1-2 hours with a peak effect occurring at 4-8 hours.[18] Goodman and Gilman describe the maximal effect being apparent 4 to 6 hours after administration.[19]

Tablet formulation can have a significant effect on absorption and hence bioavailability. Tablet ingredients include materials to hold the tablet together and to break up the tablet when consumed. Tablet ingredients can include the following:

- Drug – in this case digoxin
- Lubricant – to facilitate the tablet pressing process
- Granulating agent - tends to stick the ingredients together
- Filler – to give the tablet a size that is easy to handle
- Wetting agent - helps the penetration of water into the tablet
- Disintegration agent - helps to break the tablet apart

Inappropriate combinations of the above can result in tablets that are (1) too hard and do not break apart when consumed which results in a decrease in absorption; or (2) not hard enough and break apart too easily, increasing the exposed surface area and increasing absorption of the drug.

Niazi spoke about the effect of particle size on absorption of digoxin.[20] One study quoted by Niazi, reports that by decreasing particle size from 102*u* to between 7 and 13*u*, absorption increased to approximately 100%.[21]

The calculations, shown below, performed by Globalrph,[22] use the steady state information from Mr. McCornack quoted above. Changes in steady state levels will increase with increased bioavailability.

% Increase in Bioavailability	*Dose*	*Steady State Level*
0	0.50	1.60
10	0.55	1.76
20	0.60	1.92
30	0.65	2.08
40	0.70	2.24
50	0.75	2.40

[14] Drug Facts and Comparisons 2011, Wolter Kluwer Publisher
[15] Dorland's Illustrated Medical Dictionary, 27th Edition, Pg 206
[16] The Merck Manual, 16 Edition, 1992; Pg 456
[17] Leikin & Palouchek's, *Poisoning & Toxicology Handbook*, 3rd Edition (2002), Pg 485
[18] The Merck Manual, 16 Edition, 1992; Pg 456
[19] Gilman et al, *Goodman and Gilman's The Pharmacological Basis of Therapeutics*, 8th Edition, 1990; Pg 828
[20] Niazi S., *Textbook of Biopharmaceuticals and Clinical Pharmacokinetics*, (1979); Pg 29
[21] Journela et al, Effect of Particle Size on the Bioavailability of Digoxin (1975); *J Clin Pharmacol* 8:365
[22] http://www.globalrph.com/digoxinss.cgi

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Life threatening arrhythmias are the most important toxic effect of digoxin.[23] Clinical manifestations of digoxin toxicity include all forms of cardiac arrhythmia including ventricular premature depolarizations, junctional tachycardia and AV block among others.[24] The probability of digoxin-induced arrhythmias is as follows:[25]

Digoxin Concentration	*% probability of digoxin-induced arrhythmias*
1.7 ng/mL	10%
2.5 ng/mL	50%
3.3 ng/mL	90%

Non-cardiac symptoms of toxicity include gastrointestinal and neurophyschiatric symptoms (nausea, vomiting, diarrhea, agitation, lethargy, and visual disturbances).

The factors influencing the "likelihood of toxicity" were described in Goodman and Gilman.[26] These factors include the following:

1. incorrect selection of a maintenance dose,
2. use of a diuretic that decreases potassium,
3. patient ingesting an incorrect number of tablets,
4. increased absorption due to intestinal changes associated with antibiotic usage,
5. decrease in renal function,
6. electrolyte abnormalities
   a. depletion of potassium,
   b. abnormally high calcium levels, or
   c. abnormally low magnesium levels,
7. hypothyroidism (decreasing digoxin elimination),
8. other pharmacological agents,
9. advanced age, or
10. change in the formulation of the drug resulting in increased bioavailability,
    a. the result of an unknown change in formulation by the manufacturer, or
    b. caused by the substitution of one brand for another with different formulations

Diltiazem is a calcium channel blocking agent with the following characteristics:

- Distribution: V_d: 3-13 L/kg
- Protein binding: 70% to 80%
- Bioavailability: Oral: ~40% (undergoes extensive first-pass metabolism)

[23] The Merck Manual, 16 Edition, 1992; Pg 455
[24] *Manual of Medical Therapeutics, 26th Edition*, Department of Medicine, University of Washington School of Medicine (1989)
[25] Goodman & Gilman's The Pharmacological Basis of Therapeutics, 11th Edition , 2006
[26] Gilman et al, *Goodman and Gilman's The Pharmacological Basis of Therapeutics*, 8th Edition, 1990; Pg 834

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- Half-life elimination: Immediate release tablet: 3-4.5 hours, may be prolonged with renal impairment; Extended release tablet: 6-9 hours; Extended release capsules: 5-10 hours; I.V.: single dose: ~3.4 hours; continuous infusion: 4-5 hours
- Time to peak, serum: Immediate release tablet: 2-4 hours; Extended release tablet: 11-18 hours; Extended release capsule: 10-14 hours
- Excretion: Urine (2% to 4% as unchanged drug; 6% to 7% as metabolites); feces Absorption: Immediate release tablet: >90%; Extended release capsule: ~93%
- Maximum dose is 540mg/day.[27 28]

Diltiazem does exhibit post-mortem redistribution.[29] Calcium channel blockers have been associated with an increase in digoxin levels.[30] The American Association of Poison Control Centers practice guidelines do not list a toxic diltiazem blood level.[31]

The question of whether we can determine the level of digoxin in the blood at the time of death arises in this case. Hair analysis has not shown a correlation between blood digoxin and digoxin in hair that can be useful.[32]

Certain changes occur in the drug concentrations found in the deceased human. As the body dies, active processes that distribute a drug up a concentration gradient cease, cells die sometimes releasing their contents into the extracellular space and into the blood.[33] Diffusion can occur at all levels in the body resulting in redistribution.

Post-mortem redistribution (PMR) refers to one subset of those changes.[34] It involves the redistribution of drugs into the blood from solid organs such as the liver, lungs or, as in this case, the myocardium. The properties of a drug that one should consider when assessing the extent of PMR is the drug's volume of distribution, lipophilicity, and pKa.

Basic, highly lipophilic drugs with a volume of distribution greater than 3 L/Kg were identified by Yarema and Becher as candidates for PMR. Digoxin was identified by these authors as a candidate for PMR.[35] Some authors maintain that very high *in vivo* volumes of distribution with high concentrations in specific tissues have been proposed as a

[27] Lexicomp Online, rev. April 2011
[28] Olson et al, Calcium Channel Bloker Ingestion: An Evidence-Based Consensus Guideline for Out-of-Hospital Management, *Clinical Toxicology* (2005); 43:797-822
[29] Moriva F., Hashimoto Y., Redistribution of diltiazem in the early post-mortem period, *J Anal Toxicol* (2004); 28(4): 269-71
[30] Cardiac Glycosides/Calcium Channel Blockers (Nondihydropyridine), Lexicomp Online Interaction Monograph, April 2011
[31] Olson et al, Calcium Channel Bloker Ingestion: An Evidence-Based Consensus Guideline for Out-of-Hospital Management, *Clinical Toxicology* (2005); 43:797-822
[32] Deveaux et al, Immunoassay of digoxin in hair, *J Forensic Sci* 1997; 84: 219-223
[33] Yarema M.C. and Becker C.E., Key concepts in post-mortem drug redistribution, *Clin Toxicol (Phila)* 2005; 43(4): 236
[34] Koren G. and MacLeod S.M., Post-mortem redistribution in digoxin in rats, *J Forensic Sci*, 1985; 30(1): 92-6
[35] Yarema M.C. and Becker C.E., Key concepts in post-mortem drug redistribution, *Clin Toxicol (Phila)* 2005; 43(4): 235

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marker for PMR.[36 37 38] Ferner points out that the density of the body is approximately 1 kg/L, so that if a drug is uniformly distributed in a person weighing 70kg (the standard man), the volume of distribution will be 70L. This suggests that the volume of distribution used to determine if PMR will be present should be a volume of distribution of approximately 70L.[39]

Post-mortem redistribution (PMR) will result in a drug moving from sites of high concentration to sites of lower concentration. Ferner states:

> "Where the distribution is non-uniform before death, the possibility arises after death that the distribution will become more uniform simply because there will be flow down any concentration gradient from high concentration to lower concentration.... Organs in which drug is concentrated are loci of high concentrations, and concentrations in surrounding tissue can be disproportionately affected. The concentration measured in blood, in these circumstances, depends strongly on the sampling site. For example, digoxin is preferentially distributed to cardiac muscle, after death, concentrations in heart blood are substantially higher than those in femoral venous blood, presumably because of redistribution from cardiac muscle into heart blood."[40 41]

Digoxin is preferentially distributed to cardiac muscle. At steady state, the concentration of digoxin in the cardiac tissue is 15 to 30 times those of the plasma. The concentration in the skeletal muscle is about half that in the heart.[42]

Digoxin may exhibit a passive redistribution after death.[43] It has been suggested that the 'ideal site' to obtain blood is a ligated or clamped femoral vein.[44] Peripheral blood is less likely to have an altered digoxin level due to its distance from solid organs such as the liver, lungs or the myocardium. The femoral vein is a large vein in the groin and upper thigh. It is the primary route for the return of blood to the heart from the lower extremities. In general, redistribution into peripheral vessels, such as the femoral vein, is less than redistribution into central vessels.[45]

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[36] Pounder DJ, Jones GR, Post-mortem drug redistribution — A Toxicological Nightmare, *Forensic Sci Int* 1990; 45: 253-63

[37] Leikin J, Watson W, Post-mortem toxicology: What the Dead can and cannot tell us. *J Toxicol Clin Toxicol* 2003; 41: 47-56

[38] Hilberg et al, The extent of post-mortem drug redistribution in a rat model. *J Forensic Sci* 1999; 44: 956-62

[39] Ferner R.E., Post-mortem clinical pharmacology, *Br J of Clin Pharmacol* (2008); 66(4): 435

[40] Ferner R.E., Post-mortem clinical pharmacology, *Br J of Clin Pharmacol* (2008); 66(4): 430

[41] Holt D.W. and Benstead J.D., Post-mortem assay of digoxin by radioimmunoassay, *J Clin Pathol* (1975); 28: 483

[42] Gilman et al, *Goodman and Gilman's The Pharmacological Basis of Therapeutics*, 8th Edition, 1990, Pg 828

[43] Pelissier-Alicot et al, Mechanisms underlying post-mortem redistribution of drugs: a review, *J. of Analytical Toxicology*, 27(8): 533

[44] Yarema M.C. and Becker C.E., Key concepts in post-mortem drug redistribution, *Clin Toxicol (Phila)* 2005; 43(4): 235

[45] Cook DS, Braithwaite RA, Hale KA; Estimating ante mortem drug concentrations from post-mortem blood samples: the influence of post-mortem redistribution, *J Clin Pathol* (2000); 53:282-285

There is considerable discussion about the findings of Vorpahl and Coe in the literature.[46] The purpose of their study was to determine a way to estimate the digoxin level ante mortem from post-mortem levels. They proposed a post-mortem to ante mortem ratio of 1.96 for heart vein samples, 1.63 for subclavian vein samples, and 1.42 for femoral vein samples. Using this ratio, Mr. McCornack's post-mortem level of 3.6 ng/mL would equate to an ante mortem level of 2.5 ng/mL.

Location	*Ante/Post Ration*	*Extrapolated Blood Level*
Heart Vein	1.96	1.8
Subclavian Vein	1.63	2.2
Femoral Vein	1.42	2.5

Koren and MacLeod showed with a post-mortem analysis of rats that when the digoxin concentration is in the therapeutic or low toxic range, digoxin may re-enter the blood stream after death by passive redistribution. However, high ante-mortem digoxin concentrations "may prevent such passive redistribution." Their conclusion was that: "Therefore, ante mortem digoxin intoxication cannot be reliably inferred on the basis of high post-mortem levels of the drug." The authors continued to state that "Digoxin intoxication can be ruled out when post-mortem SDC (serum digoxin concentration) remain within the therapeutic range."[47]

When interpreting post-mortem drug levels, post-mortem drug redistribution needs to be considered.[48] Post-mortem drug levels do reveal some facts and it has been said that the integration of ante mortem history with post mortem drug concentrations can be used to glean some facts about the deceased.[49] Laboratory data obtained after death considered in light of the pre-death laboratory data correlated with the patients' clinical condition at the time of death is necessary to render an appropriate opinion on the cause of death.[50]

Based on the "likelihood of toxicity" factors described by Goodman and Gillman and the information described above, I have made the following analysis:

Mr. McCornack was not on an antibiotic and did not report any intestinal changes other than a vague bloated feeling. He was not on a potassium sparing diuretic. There is no evidence of hypothyroidism, an electrolyte abnormality or decreased renal function. And he was not of an advanced age.

Mr. McCornack had been on this drug regimen for years. At his last check up, he exhibited no signs or symptoms leading his physicians to change this drug regimen. At his last full workup by his physician, the patient had a digoxin level obtained at steady state that was appropriate for his diagnosis.

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46 Vorpahl T.E. and Coe J.L., Correlation of ante mortem and post-mortem digoxin levels, J Forensic Sci (1978); 23(2): 329-34

47 Koren G. and MacLeod S.M., Post-mortem redistribution in digoxin in rats, *J Forensic Sci*, 1985; 30(1): 92-6

48 Shepherd M.F., Lake K.D. and Kamps M.A., Post-mortem changes and pharmacokinetics: Review of the Literature and Case Report, *The Annals of Pharmacotherapy*, 1992; 26(4): 510-14

49 Kennedy MC, Post-mortem Drug Concentrations, *Intern Med J* (2010); 40(3): 183-7

50 Yarema M.C. and Becker C.E., Key concepts in post-mortem drug redistribution, *Clin Toxicol (Phila)* 2005; 43(4): 235

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Mr. McCornack was taking one 0.25mg tablet two times a day. The level obtained on May 15, 2007, from Mr. McCornack was from a blood sample obtained just before his next dose. Normally, analysis is done with peak levels obtained 4 to 8 hours (preferably at 6 hours) after administration. Mr. McCornack's level, on the other hand, was taken at a time that reflects his lowest drug level over the course of a day.

I have considered other causes for his digoxin to be elevated, including his use of diltiazem. Considering that the patient had been on this drug regimen for some time with no ill effects, nothing points to diltiazem as the causal agent on this particular night.

The last factors to consider are an incorrect administration of the evening dose or an abnormality in the tablet ingested.

It my understanding that Mr. McCornack was using a daily tablet dispenser. Mr. McCornack distributed his tablets into the dispenser for each day's dose. The possibility that Mr. McCornack would have consumed an incorrect number of tablets is highly unlikely because of this fact.

It should be noted that Mr. McCornack suffered a cardiac arrest at the time the digoxin blood level was reaching its maximum peak. We know from Mr. McCornack's post-mortem drug levels that he had a large amount of digoxin stored in his body (peripheral digoxin blood level was 3.6 ng/mL). Considering that the deceased had been stable on his medications at their current doses for some time, something was different the night he died. In my opinion, Mr. McCornack's digoxin blood level was reaching its maximum at the time of his demise and this was a primary factor in his death.

In the year prior to his death, Mr. McCornack was asymptomatic as to the effects of digoxin poisoning. If Mr. McCornack did have a high or elevated digoxin level for reasons other than a tablet abnormality, he was asymptomatic by all accounts that I could review.

Therefore, it my opinion that:

- Mr. McCornack had an elevated digoxin level at the time of his demise,
- The elevated digoxin level was probably the result of a change in the formulation of the Digitek tablet or a non-conforming tablet, and
- Judging from Mr. McCornack's clinical conditions on the night of March 23, 2008, digoxin poisoning was the cause of his death.

I appreciate the opportunity to work on this interesting fact pattern.

Sincerely.

Keith Patrick Gibson, Pharm.D., J.D.

KPG/dbm

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Other References:

1. Jusko WJ, et al. Pharmacokinetic design of digoxin dosage regimens in relation to renal function. J *Clin Pharmacol* 1974;14:525-35.
2. Koup JR, et al. Digoxin pharmacokinetics: role of renal failure in dosage regimen design. *Clin Pharmacol Ther* 1975;18:9-21.
3. Walsh FM, Sode J. Significance of non-steady-state serum digoxin concentrations. *Am J Clin Pathol* 1975;63:446-50.
4. Dobbs SM, Mawer GE. Prediction of digoxin dose requirements. *Clin Pharmacok* 1977;2:281-91.
5. Koda-Kimble MA: *Congestive heart failure, in Applied Therapeutics for Clinical Pharmacists*, 2nd ed, edited by MA Koda-Kimble et al, Applied Therapeutics, Inc, San Francisco 1978; pp 161-86.
6. Thomas RW, Maddox RR. The interaction of spironolactone and digoxin: a review and evaluation. *Ther Drug Monit* 1981;3:117-20.
7. Klein HO, et al. The influence of verapamil on serum digoxin concentration. *Circul* 1982;65:998-1003.
8. Hyneck ML, et al. Comparison of methods for estimating digoxin dosing regimens. *AJHP* 1981;38:69-73.
9. Bigger JT. The quinidine-digoxin interaction. *Mod Con Card Dis* 1982;51:73-78.
10. Lalonde RL, Pao D. Correlation coefficient versus prediction error in assessing the accuracy of digoxin dosing methods. *Clin Pharm* 1984;3:178-83.
11. Reuning RH, Garaets DR. "Digoxin", in Evans W, Schentag J, Jusko J (eds): *Applied Pharmacokinetics*. Applied Therapeutics, Inc, San Francisco 1986; pp 908-43.
12. Pounder D. and Jones G., "Post-mortem drug redistribution — A Toxicological Nightmare", *Forensic Science International*, 1990; 45(3): 253

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Compliance with Rule 26

A. Statement of Opinion

   a. See report above.

B. Facts or Data Considered

   a. See attached report and list of references
   b. Deposition of Kathy McCormack, 10-05-2009, and Exhibits
   c. Deposition of Lawrence Von Dollen, M.D., 10-05-2009, and Exhibits
   d. Deposition of Richard T. Mason, M.D., 10-01-2009, and Exhibits
   e. Deposition of Gordon Lemm, M.D., 10-02-2009, and Exhibits
   f. Expert Report of David M. Bliesner
   g. Expert Report of Dr. Walter Kernan dated July 1, 2010

C. Exhibits

- None

D. Witness Qualifications

- **Professional License**:
  - California Pharmacist License Number 35695
- **Education**
  - Doctor of Pharmacy, University of Southern California, School of Pharmacy (1980)
- **Work Experience**
  - See attached resumes.

E. List of Publications Authored in the Previous 10 years

- None

F. List of Cases in Which Expert Testified or was Deposed in the last 4 years

- None

G. Statement of Compensation. See attached Billing Rate Sheet.

Dated: May 16, 2011

Keith Patrick Gibson, Pharm.D., J.D.

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KEITH PATRICK GIBSON, PHARM.D., J.D.

Forensic Pharmaceutical Consultant

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Rates for Professional Services

- Preparation .... $250/hr.
    - Review of reports
    - Literature research for medical or forensic topics
    - Medical chart evaluation
- Court testimony or deposition .... $400/hr. (4 hrs minimum)
- Travel time .... $125/hr.
- Minimum charge for any travel outside county $2000/day
- Costs for travel and housing are billed as received.
- Costs for production of professional articles are billed as received.

Should any questions arise or to make arrangements, please contact me at the email or other address above.

Sincerely,

Keith Patrick Gibson, Pharm.D., J.D.

(effective 1-1-2011)
KPG/dbm

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KEITH PATRICK GIBSON

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EDUCATION

UNIVERSITY OF THE PACIFIC — McGeorge School of Law
*Top 40% of Class — **JURIS DOCTOR** — December 1985

UNIVERSITY OF SOUTHERN CALIFORNIA — School of Pharmacy
Graduation Award: Obergfel Brothers Award — **DOCTOR OF PHARMACY** — June 1980

PROFESSIONAL LICENSES

California Bar Number 127278
Passed: February 1986 Exam (First Attempt)
Admitted: December 1986

California Pharmacist #35695

United States District Court
Eastern District of California
Admitted: December 1986

EMPLOYMENT

Maguire & Ashbaugh — Position: **Deputy Public Defender**
991 Osos Street, Suite A, San Luis Obispo California 93401 — July 1987 to Present
Worked under contract for the law firm of Maguire & Ashbaugh, who have the primary contract to perform the services of the Public Defender for the County of San Luis Obispo, California. While under contract, performed all the duties of a Deputy Public Defender with total responsibility for all cases at all phases from arraignment through trial and sentencing in the assigned Department of the Superior Court.

Office of Administrative Hearings — Position: **Administrative Law Judge *pro tempore***
501 J Street, Suite 230, Sacramento, California 95814 — March 1995 to Present
Served under contract as a pro tempore Administrative Law Judge to preside over hearings for the Office of Administrative. Hearings concerning the authorization for the involuntary administration of psychotropic medications by the California Department of Corrections & Rehabilitation (CDCR) to individuals confined within the jurisdiction of the CDCR. Conducted assigned proceedings and made all orders prior to, during and at the conclusion of the hearing.

Forensic Pharmaceutical Consultant — Position: **Consultant & Expert Witness**
1241 Johnson Avenue #318, San Luis Obispo, California 93401 — August 1988 to Present
Provided information to lawyers about the pharmacology of both legal and illegal drugs. Provided information about the physiology of the human organism including the physiology of the kidney and liver as it relates to the detection and elimination of drugs. Conducted investigations, prepared reports and testified as an expert witness.

Private Practice — Position: **Attorney**
1108 Garden Street, Suite 205, San Luis Obispo, California 93401 — December 1990 to November 1994
Past responsibilities with the Public Defender allowed for a non criminal law private practice that results in a total of about 5 to 10 hours of a work week spent on civil law matters. Handled cases involving workers' compensation, family law, debt collection, unlawful detainer and general business law issues.

Marian Medical Center — Position: Clinical/Operational **Staff Pharmacist**
1400 Church Street, Santa Maria, California — August 2005 to Present
Currently working as a clinical/operational staff pharmacist, responsible for all aspects of the pharmacy operation including clinical services, drug information, patient chart review, order entry, IV admixture and technician supervision.

Sutter Community Hospitals — Position: **Staff Pharmacist**
52nd and F Streets, Sacramento, CA 95831 — July 1981 to July 1987
Staff pharmacist duties included the following: inpatient and outpatient orders, maintained patient profiles, IV admixture program, control of aseptic technique and admixture incompatibilities, unit does system, chemotherapy admixture program, quality control audits, technician supervision and information retrieval for medical staff. Developed Aseptic Technique Course and manual. Wrote orientation manual and orientation procedures for new employees.

ORGANIZATIONS

President, Cuesta Society of Hospital Pharmacists (1991)
San Luis Obispo County Bar Association — American Association for the Advancement of Science (AAAS)
American Civil Liberties Unions (ACLU) — California Attorneys for Criminal Justice (CACJ)

CLAIM TO FAME: Football: Honorable mention, All League (1973 - College of the Sequoias), attended University of Hawaii on sports scholarship (1974). (Rev. 2011-01)

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Education

1980 – **Doctor of Pharmacy**, University of Southern California, School of Pharmacy

1985 – Juris Doctor, University of the Pacific, McGeorge School of Law

Experience

2005 – Present Clinical Pharmacist, **Marian Medical Center**, Santa Maria

Currently working as a clinical staff pharmacist, responsible for all aspects of the pharmacy operation including clinical services, drug information, patient chart review and drug distribution.

2004 – 2005 Staff Pharmacist, **Community Health Centers** of the Central Coast, San Luis Obispo Pharmacy

Worked the out-patient pharmacy, participated in the services provided to the Mental Health In-Patient Unit which includes the delivery of unit dose, Pryxis, QA (quality assurance), DUR (drug utilization review), policy and procedure development and on-call pharmacist services.

1992 – 2004 Staff Pharmacist, **San Luis Obispo County Hospital**

Worked in the in-patient and out-patient pharmacies. Participated in the daily dispensing of drugs. Duties included dispensing as well as those usually assigned to a clinical pharmacist. Participated in QA (quality assurance) and DUE (drug utilization review).

1990 – 1992 Staff Pharmacist, **Marian Medical Center**

1987 – 1990 Staff Pharmacist, **Twin Cities Community Hospital**

Worked in the in-patient pharmacy and participated in pharmacist controlled drug protocols.

1981 – 1987 Staff Pharmacist, **Sutter Community Hospital**

Staff pharmacist duties included the following: in-patient and out-patient orders, maintained patient profiles, IV admixture program (with Harvard infusion pumps), control of aseptic technique and admixture incompatibilities, unit does system, chemotherapy admixture program, quality control audits, inventory control, prepackaging program, technician supervision and information retrieval for medical staff. Developed an Aseptic Technique Course and manual. Wrote the orientation manual and orientation procedures for new employees.

References

Cliff Elliott, former Director of Pharmacy, San Luis Obispo County General Hospital
Additional references available upon request

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Forensic Pharmaceutical Consultant

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